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Head Injury and Combat-Related Posttraumatic Stress Disorder

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Given the association of injury and posttraumatic stress disorder (PTSD), we examined whether head injury might be associated with increased frequency and severity of PTSD. Using a mail survey, we queried 143 male combat veterans with and without PTSD, who had previously participated in PTSD research in our laboratory, about their history of head injury. Respondents with a PTSD diagnosis were significantly more likely to report a history of head injury than those without. Patients with a history of head injury also reported more severe symptoms of PTSD compared with PTSD patients without head injury. The association of head injury and PTSD was not due to greater combat exposure in the head-injured group. Head injury is associated with a greater likelihood of developing combat-related PTSD and with more severe PTSD symptoms. This retrospective study did not address mechanisms that could account for this finding. The results indicate head injury should be systematically assessed by both nonpsychiatric and psychiatric physicians concerned with the psychological sequelae of exposure to victimizing experiences.

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The environmental events that can give rise to posttraumatic stress disorder (PTSD; Fairbank et al., 1993) are often associated with violence and injury. This association suggests the importance of examining the effects of head injuries on the development of PTSD because this type of injury can affect the central nervous system. Although there is increased interest in the neurocognitive aspects of PTSD as reflected in studies examining intelligence (Dalton et al., 1989), neuropsychological test performance (Wolfe and Charney, 1991), neurological status (Gurvits et al., 1993), and neuroanatomical changes (Bremner et al., 1995; Semple et al., 1993), the possible association of head injury and PTSD has been largely neglected.

The sequelae of mild head injury have significant, though often unrecognized, influences on cognitive and neuropsychological functioning (Leininger and Kreutzer, 1992), psychiatric disability, and family/social adaptation (Andrasik and Wincze, 1994). Affective and personality changes are also associated with closed head injuries (Slagle, 1990) and may result in psychiatric disorders (Fann et al., 1995). Depression is the leading psychological symptom of mild head injury (Andrasik and Wincze, 1994), though anxiety is also common (Evans, 1994).

Horton (1993) and King (1997) have each provided case study evidence of the co-occurrence of mild head injury and PTSD, while Grisby and Kaye (1993) reported high comorbidity rates between PTSD and vertigo (a common symptom after head injury) in a large sample of head-injured individuals. Wright and Telford (1996) reported that 50 head-injured participants showed signs of PTSD and psychological distress 6 months and 3 years postinjury. Although some authors have presented data in support of the proposition that posttraumatic amnesia is incompatible with the development of PTSD (Sbordone and Liter, 1995; Warden et al., 1997), others have reported that PTSD may develop despite loss of consciousness or amnesia for the traumatic event (Bryant, 1996; Layton and Wardi-Zonna, 1995; McGrath, 1997; McMillan, 1991, 1996), though these latter reports are case studies. Ohry et al. (1996) evaluated 24 patients for PTSD symptoms by using paper and pencil measures and found that 33% of the patients met criteria for PTSD. However, none of these studies used structured interview methods to carefully diagnose PTSD in large samples, nor did they employ a control group to assess whether the

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risk for PTSD or its severity is increased by head injury.

The present study a) compared the frequency of head injury in combat veterans with and without PTSD, b) evaluated whether self-reported head injury is associated with the severity of PTSD, and c) examined the sites, causes, and symptoms of head injury reported by veterans with PTSD.

Methods

Participants

There were a total of 350 potential respondents. All were male combat veterans who had served in the United States military. Participants were originally recruited for studies of PTSD conducted over the past 5 years. The original studies addressed the relationships between PTSD and anger, PTSD and cognitive processing, and psychophysiological aspects of the disorder. Participants in the prior studies included combat veterans with and without a PTSD diagnosis. Veterans were recruited through newspaper advertisements, posters, announcements to community organizations, VA psychiatric clinics, local Vet Centers, and through mailings to patients attending the medical outpatient clinics of the local VA Center.

The majority of the participants underwent careful psychiatric diagnosis, comprising psychometric measures and structured clinical interviews, either the Structured Clinical Interview for DSM-III-R, Nonpatient, Vietnam version (SCID-NP-V; Spitzer et al., 1989), or the Clinician Administered PTSD Scale (CAPS; Blake et al., 1990). The SCID-NP-V was used to measure the presence and absence of PTSD and to determine the presence of other psychiatric disorders. The CAPS, a more detailed measure, was used to measure the presence or absence of PTSD instead of the PTSD module of the SCID-NP-V. However, for some participants, the remaining modules of the SCID were used to evaluate other psychiatric diagnoses. Veterans meeting DSM-III-R (American Psychiatric Association, 1989) criteria for a diagnosis of organic mental disorder, psychotic disorder, current (within the past 30 days) substance abuse or dependence, or antisocial personality disorder were excluded from the parent studies as well as from the present study.

Procedure

Survey packets were mailed to participants with a cover letter explaining the purpose of the study as well as the confidential and voluntary nature of participation. All participants had signed written consents to participate in studies in this laboratory. Participants were explicitly informed in a cover letter that declining participation in the survey would not affect their benefits or continued treatment. Therapists treating veterans with PTSD in the VA were informed of the survey. Patients were not compensated for participation in the study.

Reminder letters were sent to those who did not respond to the packet, approximately 6 to 8 weeks after the initial mail-out. If no response to the reminder letter was received, a second packet was sent out, approximately 6 weeks later. Packets comprised the following instruments:

Head Injury Questionnaire. Head injury history was assessed with a 7-item self-report questionnaire. If the respondent indicated experiencing a head injury in the past, he was asked to report on the most severe head injury sustained in his lifetime. Head injury questionnaire items assessed injury location (*i.e.*, on the head), cause of injury (*e.g.*, automobile accident, fight, etc.), symptoms (*e.g.*, loss of consciousness, nausea, headaches, etc.), age at the time of injury, and whether medical treatment was sought for the injury. Finally, respondents were asked to report the total number of head injuries they had sustained over their lifetime.

Combat Exposure Scale (CES). The CES is designed to assess wartime stressors and provides a continuous index of the intensity and severity of combat. Keane et al. (1989) reported good internal consistency (coefficient alpha = .85) and reliability (1-week test-retest reliability = .97). Veterans with PTSD consistently score significantly higher than a comparison group of Vietnam combat veterans without PTSD (20).

Mississippi Scale for Combat-Related PTSD. The Mississippi Scale (Keane et al., 1988) is a continuous measure of PTSD symptoms and of its associated features. It can also be used as a categorical measure to index the presence or absence of a PTSD diagnosis. Keane et al. (1988) reported a sensitivity of .93 and a specificity of .89, and an overall efficiency of .90 in differentiating veterans with and without PTSD using a cut-off score of 107. Very strong support for the Mississippi Scale as a categorical measure of combat-related PTSD was provided by the landmark National Vietnam Veterans Readjustment Study (Kulka et al., 1990).

Results

Participants

Response Rate. We calculated the response rate using Babbie's method (1973). Of the total 350 potential respondents, 6 were deceased, and 63 were

TABLE 1
Means and Percentages of Demographic and Psychometric Measures of Head-Injured (HI) and Non-Head-Injured Veterans (NHI) by PTSD Status

	HI		NHI	
	Non-PTSD	PTSD	Non-PTSD	PTSD
Demographic Data				
Age (yr)	51.13	49.55	52.70	47.81
	(7.51)	(7.60)	(8.13)	(5.58)
Education (yr)	15.00	13.36	15.38	13.31
	(2.15)	(2.46)	(2.55)	(1.70)
Ethnicity				
White	48.6%	41.7%	45.8%	35.7%
	(17)	(20)	(11)	(5)
Nonwhite	51.4%	58.3%	54.2%	64.3%
	(18)	(28)	(13)	(9)
Marital status				
Married	22.9%	28.1%	41.9%	20.9%
	(22)	(27)	(18)	(9)
Divorced	8.3%	20.8%	7.0%	9.3%
	(8)	(20)	(3)	(4)
Never married	5.2%	18.5%	13.9	7.0%
	(5)	(13)	(6)	(3)
Psychometric data				
CES	21.57	25.70	18.36	25.25
	(7.19)	(6.26)	(6.54)	(7.92)
Mississippi	85.84	131.48	75.93	124.31
	(15.17)	(13.37)	(18.64)	(12.47)

Numbers in parentheses are standard deviations for the interval-level variables (age, education, Mississippi, and CES) and frequencies for the nominal variables.

undeliverable due to address problems and were excluded from consideration. Of the remaining 281 subjects, 143 responded to the mail-out. This represents a response rate of 51%, which compares favorably with mail surveys of psychiatric patients (Baradell, 1995; Creamer et al., 1996).

Comparing Responders with Nonresponders. A strength of the present design is that we could evaluate the possible impact of response biases introduced by responder self-selection because relevant data were obtained in the course of the veterans' prior participation in research. We compared responders with nonresponders on age, marital status, branch of service, education, ethnicity, Mississippi Scale scores, and combat exposure. Survey responders were older ($t[340] = 2.55$, $p < .012$) and were less likely to be divorced (χ^2 [2, $N = 330$] = 6.94, $p < .04$) than nonresponders. However, responders did not differ from nonresponders on the key indices of Mississippi Scale scores ($t[279] = .28$, $p > .77$) and combat exposure ($t[297] = .20$, $p > .84$). Nor did they differ on education level, ($t[315] = 1.45$, $p > .14$) or on branch of service (χ^2 [2, $N = 328$] = .26, $p > .99$). The proportion of white to nonwhite (*i.e.*, native/part-native American, Asian, black, and Hispanic) veterans was similar for both groups (χ^2 [1, $N = 335$] = 1.56, $p > .21$).

Sample Representativeness. To evaluate sample representativeness, we compared responders whose scores on the Mississippi Scale exceeded 106 (indicating PTSD, Keane et al., 1988) with 134 treatment-seeking veterans drawn from consecutive outpatient admissions to a specialized outpatient PTSD treatment unit whose scores on the Mississippi Scale also exceeded 106. Our responder sample ($N = 77$) did not differ from the help-seeking group ($N = 100$) on Mississippi Scale scores ($t[175] = .08$, $p > .93$) and ethnicity (white *vs.* nonwhite; χ^2 [1, $N = 154$] = .15, $p > .69$). Responders with Mississippi Scale scores greater than 106 were significantly older ($t[175] = 4.29$, $p < .001$) and had lower rates of divorce (χ^2 [2, $N = 172$] = 11.08, $p < .005$), than the outpatient help-seeking group. This comparison indicates that our group was not different on the key variables of combat exposure, Mississippi Scale scores, and ethnicity from the help-seeking sample whose scores were in the PTSD range.

We conducted a similar comparison of responders scoring less than 107 on the Mississippi Scale ($N = 66$) to the corresponding help-seeking veterans scoring less than 107 ($N = 34$). Our non-PTSD scoring sample had a greater proportion of married veterans (X^2 [2, $N = 95$] = 6.25, $p < .05$) as well as a trend toward being older ($t[97] = 1.94$, $p < .06$) than the corresponding help-seeking veterans. The two groups did not differ on Mississippi Scale scores ($t[98] = 1.41$, $p > .16$). The proportion of white to nonwhite veterans was approximately 50% for each group (χ^2 [1, $N = 90$] = .02, $p > .88$).

Participant Characteristics by Head Injury and PTSD Status. To evaluate the relationship between PTSD status and head injury status, we conducted separate 2 (head injured/nonhead injured) × 2 (PTSD/non-PTSD) ANOVAs for age, combat exposure, education, and Mississippi Scale total scores. The results of all ANOVAs showed a significant effect for PTSD/non-PTSD: age ($F[1,136] = 5.23$, $p < .025$); CES ($F[1,137] = 18.71$, $p < .0001$); education ($F[1,136] = 17.06$, $p < .0001$); and Mississippi Scale scores ($F[1,137] = 279.52$, $p < .0001$).

There were no significant differences due to head injured/nonhead injured status on age, combat exposure, or education. However, head-injured participants had significantly higher total Mississippi Scale scores ($F[1,137] = 9.22$, $p < .004$) than did veterans reporting no head injuries. There were no differences among the four groups with respect to ethnicity (white *vs.* nonwhite) and marital status. Table 1 presents demographic data broken down into head injured/nonhead injured by PTSD/non-PTSD.

TABLE 2
Percentage of Locations, Causes, and Symptoms of Head Injuries and Associated Mississippi Scale Scores

	Percentage	Mississippi
Location		
Front	32.5	111.85
Back	22.9	125.00
Top	21.7	110.39
Right	25.3	112.90
Left	18.1	118.93
Other	6.0	124.20
Cause		
Vehicular accident	15.1	117.61
Fight	19.7	126.35
Sports-related	10.5	106.44
Accident	18.6	95.81
Other	36.0	113.51
Symptoms		
Loss of consciousness	48.2	111.39
Nausea	25.9	116.54
Vision problems	42.4	121.75
Headaches	72.9	116.43
Temporary memory loss	28.2	122.96
Permanent memory loss	4.7	99.75
Physical problems	17.6	121.73
Other	15.3	123.08

Percentages for location and symptoms exceed 100%, due to multiple endorsements of locations and/or symptoms; Mississippi scores are different from those obtained from the analyses, due to multiple endorsements.

Head Injury

Ninety-seven of 143 respondents (68%) indicated they sustained at least one head injury during their lifetime. Of these 97, 60% ($N = 52$) also had PTSD, as assessed by Mississippi Scale scores greater than 106 (Keane et al., 1988). Data from 10 subjects in the HI group were excluded from statistical analyses because of incorrect responding on the head injury questionnaire (*i.e.*, reported on experiences of more than one head injury to questionnaire items). Table 2 presents the percentages of locations, causes, and symptoms reported by our sample, as well as associated Mississippi Scale scores.

A qualitative analysis indicated that approximately 51.6% of "other" causes were combat-related events. The most frequently reported symptoms of head injury were headaches (72.9%), loss of consciousness (48.2%), and vision problems (*e.g.*, blurring, diplopia; 42.4%). These were not trivial head injuries as a number of respondents commented on the persistence of their symptoms years after sustaining the head injury.

Head Injury and PTSD Diagnosis. The most conservative way to evaluate the relationship of PTSD diagnosis to head injury is to compare participants for whom structured clinical diagnoses (using either the SCID or CAPS) were available and which were concordant with current Mississippi Scale scores. The current Mississippi Scale score and the initial structured diagnosis should be concordant to provide a check for the presence of current PTSD. Participants who had not been administered structured diagnostic interviews as well as veterans whose current Mississippi Scale scores were not concordant with their clinical diagnosis were excluded from this particular analysis. In this subsample of 86 veterans, 68.6% ($N = 59$) reported sustaining a head injury, and 54.7% ($N = 47$) were diagnosed with PTSD. Participants reporting a head injury were more likely to have PTSD (χ^2 [1, $N = 86$] = 3.07, $p = .04$). Sixty-one percent (36 of 59) of those with head injury had PTSD compared with 40.7% (11 of 27) for those not reporting head injury.

Given that a structured clinical diagnosis was not available for all participants, we examined the relationship of head injury to PTSD in the total sample by using the diagnostic cutoff of 107 on the Mississippi Scale to define the presence or absence of PTSD (Keane et al., 1988). The presence of a head injury was significantly associated with the Mississippi Scale–derived diagnosis of PTSD (χ^2 [1, $N = 141$] = 7.92, $p < .006$). Participants with a head injury also had significantly higher Mississippi Scale scores, denoting more severe PTSD symptoms in the head injured group (t[139] = 2.87, $p < .006$) compared with the nonhead injured veterans (see Table 1).

Head Injury and Combat Exposure. It is likely that the probability of any injury, including head injury, increases as a function of greater combat exposure. To evaluate whether the association of head injury with PTSD was merely due to greater combat exposure, we conducted an ANCOVA with head injury status as the independent variable, the Mississippi Scale score as the dependent variable, and combat exposure as measured by the CES as the covariate. As expected, given the association of PTSD and the CES (Keane et al., 1989), combat exposure contributed significantly to the Mississippi Scale score (F[1,138] = 18.40, $p < .001$). However, the main effect of head injury remained significant (F[1,138] = 11.83, $p = .001$) after the influence of combat exposure was statistically controlled for. Thus, although combat exposure was significantly associated with the Mississippi Scale measure of PTSD, the association of head injury and PTSD symptoms remained even after partialing out combat exposure.

Head Injury and Comorbid Diagnoses. For those veterans for whom the SCID was available ($N = 74$), we were also able to examine the effect of comorbid

diagnoses on head injury and Mississippi Scale scores by using a 2 × 2 ANOVA in which head injury status and presence/absence of comorbid diagnoses were the independent variables and Mississippi Scale scores were the dependent variable. The results of this analysis show that head injury and comorbidity each have an independent significant effect on Mississippi scores ($F[1,70] = 11.96$, $p < .002$ and $F[1,70] = 29.17$, $p < .0009$, respectively).

Timing of Head Injury and PTSD. We were able to identify a total of 66 veterans whose records provided information about whether their head injury was sustained before, during, or after the subject's tour in the combat theater. The results of a one-way ANOVA revealed significant differences among the three groups with respect to Mississippi Scale scores ($F[2,63] = 3.26$, $p < .05$). Bonferroni-corrected post-hoc analyses revealed that the veterans who sustained their injuries during combat duty had higher Mississippi Scale scores (mean = 118.67, SD = 24.11) compared with those who experienced head injury before and after duty (mean = 99.71, SD = 26.22 and mean = 106.94, SD = 27.79, respectively).

Head Injury Characteristics and PTSD. In further analyses, we sought to assess the effects of specific head injury characteristics (*i.e.*, site, cause, and symptoms) on PTSD diagnosis and severity as measured by the Mississippi Scale. To evaluate the effect of the site of the head injury on PTSD severity, a one-way ANOVA was computed. Because some individuals reported more than one head location being affected in the injury, a "multiple locations" category was created. This was necessary to preserve the independence of data because individuals could have, otherwise, appeared in more than one category. Thus, we compared individuals with a single head injury site with each other, with those reporting multiple locations for their head injury, and with those individuals reporting no head injury. The results revealed a significant main effect for head injury location on Mississippi Scale scores ($F[7,119] = 2.80$, $p < .01$). Bonferroni-corrected contrasts revealed a difference in PTSD severity between the nonhead injured (mean = 93.52, SD = 28.75) and the "multiple locations" (mean = 122.21, SD = 21.78) groups. Other head injury locations did not differ significantly from each other or from the nonhead injured group.

Cause of head injury differentiated specific subgroups within the head injured sample as well. A one-way ANOVA revealed a significant main effect of cause of injury on severity of PTSD ($F[5,124] = 4.76$, $p < .001$). Bonferroni-corrected post-hoc analyses indicated head injuries due to fights resulted in more severe PTSD (mean = 126.35, SD = 20.90) than only head injuries due to nonvehicular accidents (mean = 95.81, SD = 28.84) and compared with the nonhead injured group (mean = 94.76, SD = 29.82).

To evaluate whether number of symptoms was associated with increased Mississippi Scale scores, we conducted a one-way ANOVA that revealed a significant main effect of number of head injury symptoms ($F(5,123) = 4.45$, $p < .001$) on Mississippi Scale scores. Bonferroni-corrected post-hoc analysis revealed significant differences in Mississippi Scale scores between the nonhead-injured sample and head-injured individuals reporting two and four symptoms (NHI: mean = 93.52, SD = 28.75; 2-symptoms: mean = 115.68, SD = 27.76; 4-symptoms: mean = 122.76, SD = 25.65, respectively).

Head Injury Severity and PTSD. To reduce respondent variability in the reporting of head injuries, we set seeking medical treatment as a minimum criterion for presence of head injury. We performed a *t*-test to examine the relationship between Mississippi Scale scores and head injury, comparing respondents who met the criterion of having a head injury that caused them to seek medical treatment with veterans without any head injury. The groups differed significantly on their Mississippi Scale scores ($t[104] = 3.43$, $p < .002$).

We also conducted a similar analysis using as our criterion for severity whether the head-injured veteran reported a loss of consciousness or amnesia for the event. The participants meeting this criterion had significantly higher Mississippi Scale scores than nonhead injured veterans ($t[91] = 3.66$, $p < .0009$). Thus, the association of head injury and PTSD cannot be attributed to a generalized response bias by PTSD patients for item endorsement. It has been proposed that loss of consciousness and PTSD do not co-occur because of interference with memory for the traumatic event (Sbordone and Liter, 1995; Warden et al., 1997). Consequently, we examined the Mississippi Scale scores of those respondents who reported suffering a loss of consciousness (mean = 112.85) versus those who had not (mean = 115.79). The two groups did not differ with respect to Mississippi Scale scores ($t[93] = .53$, $p > .6$).

The prior analyses suggest that severity of head injury may be associated with PTSD severity as indexed by the Mississippi Scale. Although our study design did not provide for a clinical assessment of head injury severity, a number of items on the questionnaire likely to be associated with severity of head trauma were examined. Specifically, reporting multiple head locations, reporting multiple symp-

toms after head injury, and whether medical treatment was sought appeared likely to be related to increased severity of head injury on the basis of the prior analyses and face validity. We therefore calculated an ad-hoc severity index for each head-injured individual by adding the total number of locations, total number of symptoms, and a 1 if medical treatment was sought or 0 if not. The range of severity index scores was 1 to 9, with a mean of 4.3 and standard deviation of 1.7. A Pearson product-moment correlation coefficient calculated for head injury severity and Mississippi Scales for head-injured participants ($N = 81$) was significant ($r = .27$, $p < .015$).

Discussion

Head injury was significantly associated with PTSD diagnosis and with increased symptom severity in our sample of combat veterans. This association was significant even when combat exposure and comorbidity were statistically controlled for. Moreover, because the Mississippi measure of PTSD can be used as a continuous measure of PTSD severity, we were able to evaluate the association of head injury with PTSD severity. Participants with head injuries reported significantly higher symptom levels as indexed by the Mississippi Scale.

Also, increased severity of head injury was related to increased severity of PTSD. The presence of multiple head trauma locations, injuries due to fights, and reporting multiple symptoms due to the head injury all were associated with higher PTSD scores. A derived severity index was correlated with Mississippi score levels. This association suggests that as head injury severity increased so did PTSD severity. The present data do not support the proposition that loss of consciousness or amnesic symptoms are incompatible with developing PTSD and are consistent with the studies by Bryant (1996), Layton and Wardi-Zonna (1995), McGrath (1997), and McMillan (1991, 1996), though these latter reports are case studies. Furthermore, the very high prevalence (68%) of head injury in our sample suggests a high risk for injury in this group of men. Veterans reporting a history of head injury were 1.67 times more likely to have a diagnosis of PTSD than those without a history of head injury.

There are a number of strengths and limitations to consider in the present study. We were able to evaluate nonresponder bias due to the availability of data on both responders and nonresponders. Also, we were able to compare our sample to help-seeking veterans with and without PTSD. These comparisons suggest that our relatively large sample was not biased on the key variables of PTSD and combat exposure. However, this study is retrospective and depends on self-report for the head injury data. Moreover, the present report addresses only the co-occurrence of PTSD symptoms with head injury. No causal inferences can be made as to whether head injury affects the development of PTSD or is simply correlated with it due to an association with a third variable. For example, a propensity to risk taking may account for the increased risk of both head injury and PTSD. Further, an important limitation of this study is that it could not conclusively address whether the head injury and the key traumatic event occurred at the same time. Finally, the design of this study did not allow us to ascertain mechanisms whereby PTSD symptoms, trauma exposure, and head injury might interact to give rise to PTSD.

It may be useful, nevertheless, to speculate about the relationship between head injury and PTSD. Information processing models of PTSD posit that PTSD arises because of inadequate processing of the emotions stimulated by the traumatic event (Chemtob et al., 1988; Foa et al., 1989) and because of continued maladaptive cognitive processing of current events. The implied assumption is that the hypothesized disturbances in information processing are due to psychopathological processes. These models generally assume an intact nervous system capacity. The present data challenge that assumption and advocate a broader biopsychological context for the understanding of PTSD. If it is true that information processing in PTSD is disturbed, it may be that the disruption in cognition is due in part to central nervous system injury (*cf.*, Gurvits et al., 1993) or to a combination of psychopathology and central nervous system injury.

The timing of the head injury is likely to imply different causal relationships. For example, having head injury before trauma exposure may increase the risk of further exposure to traumatic events because of poor risk management. Alternately, head injury before trauma may reduce the capacity of the individual to absorb the subsequent emotional processing load imposed by the trauma. Experiencing a peritraumatic head injury may interfere with a wide variety of processes involved in resolving a psychological trauma. For example, memory consolidation or event processing may be interfered with in such a way as to defeat successful resolution of the experience. Finally, posttraumatic head injury may cause the disinhibition of regulatory processes that kept the person's psychological reactions in balance. The present results support exploring these alternative conceptualizations of

PTSD and may provide, through careful composition of groups of patients with similar head injuries, an ancillary method to study brain function and PTSD.

The present study is the first large-scale empirical investigation of the relationship between head injury and PTSD. Previous reports were derived from case studies or based on relatively small samples. Thus, this study provides strong support for routine screening of patients with PTSD for head injury. It would be also important to screen patients presenting primarily with symptoms of head injury for PTSD. Research on treatment would do well to include head injury as one variable to explore because patients with head injuries may be more at risk for cognitive difficulties that might interfere with psychosocial treatment effectiveness.

Among important remaining questions is whether the association between head injury and PTSD found in this combat-exposed sample will also characterize groups exposed to other types of traumatic events. The present study examined this association only in male subjects. Thus, additional research is also needed to evaluate whether the increased risk for PTSD is also to be found in women. To best address these questions, future investigations should place particular emphasis on prospective longitudinal research in an effort to determine the timing and the mechanisms underlying this relationship. Our study assessed PTSD using thorough diagnostic methods, however our self-report method of measuring head injury is a clear limitation. Further research would benefit from including modern methods of characterizing head injury such as MRI and PET together with thorough neuropsychological evaluations of function. Obtaining such additional data would illuminate clinical and theoretical questions regarding both head injured and PTSD populations.

References

American Psychiatric Association (1989) *Diagnostic and statistical manual of mental disorders* (3rd ed rev). Washington, DC: Author.

Andrasik FA, Wincze JP (1994) Emotional and psychosocial aspects of mild head injury. *Semin Neurol* 14:60–66.

Babbie ER (1973) *Survey research methods*. Belmont, CA: Wadsworth.

Baradell JG (1995) Clinical outcomes and satisfaction of patients of clinical nurse specialist in psychiatric-mental health nursing. *Arch Psychiatr Nurs* 9:240–250.

Blake D, Weathers F, Nagy L, Kaloupek D, Klauminzer G, Charney D, Keane T (1990) *Clinician-administered PTSD scale (CAPS)*. Washington, DC: National Center for Posttraumatic Stress Disorder.

Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Southwick SM, Delaney RC, McCarthy G, Charney DS, Innis RB (1995) MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *Am J Psychiatry* 152:973–981.

Bryant RA (1996) Posttraumatic stress disorder, flashbacks and pseudomemories in closed head injury. *J Trauma Stress* 9: 621–630.

Chemtob CM, Roitblat HL, Hamada RS, Carlson JG, Twentyman CT (1988) A cognitive action theory of post-traumatic stress disorder. *J Anxiety Dis* 2:253–275.

Creamer M, Jackson A, Ball R (1996) A profile of help-seeking Australian veterans. *J Trauma Stress* 9:569–576.

Dalton JE, Pederson SL, Ryan JJ (1989) Effects of post-traumatic stress disorder on neuropsychological test performance. *Int Clin Neuropsychol* 1:121–124.

Evans RW (1994) The postconcussion syndrome: 130 years of controversy. *Semin Neurol* 14:32–39.

Fairbank JA, Schlenger WE, Caddell JM, Woods MG (1993) Posttraumatic stress disorder. In PB Sutker, HE Adams (Eds), *Comprehensive handbook of psychopathology* (2nd ed, pp 145–165). New York Plenum Press.

Fann JR, Katon WJ, Uomoto JM, Esselman PC (1995) Psychiatric disorders and functional disability in outpatients with traumatic brain injuries. *Am J Psychiatry* 152:1493–1499.

Foa EB, Steketee G, Olasov-Rothbaum B (1989) Behavioral/cognitive conceptualization of posttraumatic stress disorder. *Behav Ther* 20:155–176.

Grisby JP, Kaye K (1993) Incidence and correlates of depersonalization following head trauma. *Brain Inj* 7:507–513.

Gurvits TV, Lasko NB, Schachter SC, Kuhne AA, Orr SP, Pitman RK (1993) Neurological status in Vietnam veterans with chronic posttraumatic stress disorder. *J Neuropsychiatry Clin Neurosci* 5:183–188.

Horton AM (1993) Posttraumatic stress disorder and mild head trauma: Follow-up of a case study. *Percept Mot Skills* 76:243–246.

Keane TM, Caddell JM, Taylor KL (1988) Mississippi scale for combat-related post-traumatic stress disorder: Three studies in reliability and validity. *J Consult Clin Psychol* 56:85–90.

Keane TM, Fairbank JA, Caddell JM, Zimering RT, Taylor KL, Mora CA (1989) Clinical evaluation of a measure to assess combat exposure. *Psychol Assess J Consult Clin Psychol* 1: 53–55.

King NS (1997) Post-traumatic stress disorder and head injury as a dual diagnosis: "Islands" of memory as a mechanism. *J Neurol Neurosurg Psychiatry* 62:82–84.

Kulka RA, Schlenger WE, Fairbank JA, Hough RL, Jordan BK, Marmar CR, Weiss DS (1990) *Trauma and the Vietnam War generation: Report of findings from the National Vietnam veterans readjustment study*. New York: Bruner/Mazel.

Layton BS, Wardi-Zonna K (1995) Posttraumatic stress disorder with neurogenic amnesia for the traumatic event. *Clin Neuropsychol* 9:2–10.

Leininger BE, Kreutzer JS (1992) Neuropsychological outcome of adults with mild traumatic brain injury: Implications for clinical practice and research. *Phys Med Rehabil* 6:169–182.

McGrath J (1997) Cognitive impairment associated with posttraumatic stress disorder and minor head injury: A case report. *Neuropsychol Rehabil* 7:231–239.

McMillan TM (1991) Post-traumatic stress disorder and severe head injury. *Br J Psychiatry* 159:431–433.

McMillan TM (1996) Post-traumatic stress disorder following minor and severe closed head injury: 10 single cases. *Brain Inj* 10:749–758.

Ohry A, Rattok J, Solomon Z (1996) Post-traumatic stress disorder in brain injury patients. *Brain Inj* 10:687–695.

Sbordone RJ, Liter JC (1995) Mild traumatic brain injury does not produce post-traumatic stress disorder. *Brain Inj* 4:405–412.

Semple WE, Goyer P, McCormick R, Morris E, Compton B, Muswick G, Nelson D, Donovan B, Leisure G, Berridge M, Miraldi F, Schulz SC (1993) Preliminary report: Brain blood

flow using PET in patients with posttraumatic stress disorder and substance-abuse histories. *Biol Psychiatry* 34: 115–118.

Slagle DA (1990) Psychiatric disorders following closed head injury: An overview of biopsychosocial factors in their etiology and management. *Int J Psychiatry Med* 20:1–35.

Spitzer RL, Williams JB, Gibbon M (1989) *Structured clinical interview for DSM-III-R-non-patient Vietnam version (SCID-NP)*. New York: Biometrics Research Department, New York State Psychiatric Institute.

Warden DL, Labatte LA, Salazar AM, Nelson R, Sheley E, Staudenmeier J, Martin E (1997) Posttraumatic stress disorder in patients with traumatic brain injury and amnesia for the event? *J Neuropsychiatry Clin Neurosci* 9:18–22.

Wolfe J, Charney DS (1991) Use of neuropsychological assessment in posttraumatic stress disorder. *Psychol Assess* 3:573–580.

Wright JC, Telford R (1996) Psychological problems following minor head injury: A prospective study. *Br J Clin Psychol* 35: 399–412.